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Health Effects  
Assessment for 1,2-T-Dichloroethylene

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**HEALTH EFFECTS ASSESSMENT  
FOR 1,2-T-DICHLOROETHYLENE**

**U.S. Environmental Protection Agency  
Office of Research and Development  
Office of Health and Environmental Assessment  
Environmental Criteria and Assessment Office  
Cincinnati, OH 45268**

**U.S. Environmental Protection Agency  
Office of Emergency and Remedial Response  
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Washington, DC 20460**

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| 16. ABSTRACT<br><p>This document represents a brief, quantitatively oriented scientific summary of health effects data. It was developed by the Environmental Criteria and Assessment Office to assist the Office of Emergency and Remedial Response in establishing chemical-specific health-related goals of remedial actions. If applicable, chemical-specific subchronic and chronic toxicity interim acceptable intakes are determined for systemic toxicants, or <math>q_1^*</math> values are determined for carcinogens for both oral and inhalation routes.</p> <p>Sufficient data are not available at this time on trans-1,2-dichloroethylene to determine either acceptable intakes or <math>q_1^*</math>s.</p> |                                                  |                                                 |
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#### DISCLAIMER

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## PREFACE

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with 1,2-dichloroethylene. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary and reflect limited resources allocated to this project. Pertinent toxicologic and environmental data were located through on-line literature searches of the Chemical Abstracts, TOXLINE, CANCERLINE and the CHEMFATE/DATALOG data bases. The basic literature searched supporting this document is current up to September, 1984. Secondary sources of information have also been relied upon in the preparation of this report and represent large-scale health assessment efforts that entail extensive peer and Agency review. The following Office of Health and Environmental Assessment (OHEA) sources have been extensively utilized:

U.S. EPA. 1980a. Ambient Water Quality Criteria for Dichloroethylenes. Environmental Criteria Assessment Office, Cincinnati, OH. EPA 440/5-80-041. NTIS PB 83-117525.

U.S. EPA. 1982. Reportable Quantity for 1,2-Dichloroethylene. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, OHEA for the Office of Solid Waste and Emergency Response, Washington, DC.

U.S. EPA. 1983b. Hazard Profile for trans-1,2-Dichloroethylene. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, OHEA for the Office of Solid Waste and Emergency Response, Washington, DC.

The intent in these assessments is to suggest acceptable exposure levels whenever sufficient data were available. Values were not derived or larger uncertainty factors were employed when the variable data were limited in scope tending to generate conservative (i.e., protective) estimates. Nevertheless, the interim values presented reflect the relative degree of hazard associated with exposure or risk to the chemical(s) addressed.

Whenever possible, two categories of values have been estimated for systemic toxicants (toxicants for which cancer is not the endpoint of concern). The first, the AIS or acceptable intake subchronic, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval (i.e., for an interval that does not constitute a significant portion of the lifespan). This type of exposure estimate has not been extensively used or rigorously defined, as previous risk assessment efforts have been primarily directed towards exposures from toxicants in ambient air or water where lifetime exposure is assumed. Animal data used for AIS estimates generally include exposures with durations of 30-90 days. Subchronic human data are rarely available.

Reported exposures are usually from chronic occupational exposure situations or from reports of acute accidental exposure.

The AIC, acceptable intake chronic, is similar in concept to the ADI (acceptable daily intake). It is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifespan [see U.S. EPA (1980b) for a discussion of this concept]. The AIC is route specific and estimates acceptable exposure for a given route with the implicit assumption that exposure by other routes is insignificant.

Composite scores (CSs) for noncarcinogens have also been calculated where data permitted. These values are used for ranking reportable quantities; the methodology for their development is explained in U.S. EPA (1983a).

For compounds for which there is sufficient evidence of carcinogenicity, AIS and AIC values are not derived. For a discussion of risk assessment methodology for carcinogens refer to U.S. EPA (1980b). Since cancer is a process that is not characterized by a threshold, any exposure contributes an increment of risk. Consequently, derivation of AIS and AIC values would be inappropriate. For carcinogens, q<sub>1</sub>'s have been computed based on oral and inhalation data if available.

## ABSTRACT

In order to place the risk assessment in proper context, the reader is referred to the preface of this document. The preface outlines limitations applicable to all documents of this series as well as the appropriate interpretation and use of the quantitative estimates.

Information concerning toxicological consequences of 1,2-t-dichloroethylene exposure is extremely limited. Data were inadequate to estimate an AIS or AIC for either the oral or inhalation routes. A CS of 10.5 was derived based on histopathological changes in the liver and lungs of rats exposed via inhalation. This compound has not been tested for carcinogenicity. The limited mutagenicity data are negative.

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## LIST OF ABBREVIATIONS

|      |                              |
|------|------------------------------|
| ADI  | Acceptable daily intake      |
| AIC  | Acceptable intake chronic    |
| AIS  | Acceptable intake subchronic |
| BCF  | Bioconcentration factor      |
| CAS  | Chemical abstract service    |
| CS   | Composite score              |
| i.p. | Intraperitoneal              |
| ppm  | Parts per million            |
| STEL | Short-term exposure limit    |
| TLV  | Threshold limit value        |

## 1. ENVIRONMENTAL CHEMISTRY AND FATE

The relevant physical and chemical properties and environmental fate of 1,2-t-dichloroethylene (CAS No. 156-60-5) are as follows:

|                                      |                                      |
|--------------------------------------|--------------------------------------|
| Chemical class:                      | halogenated aliphatic hydrocarbon    |
| Molecular weight:                    | 96.95                                |
| Vapor pressure at 25°C:              | 324 mm Hg (Torkelson and Rowe, 1981) |
| Water solubility at 20°C:            | 6300 mg/l (Torkelson and Rowe, 1981) |
| Octanol/water partition coefficient: | 3 (estimated)                        |
| BCF:                                 | 0.5 (estimated)                      |
| Half-lives in                        |                                      |
| Air:                                 | 2.1 days (Hendry and Kenley, 1979)   |
| Water:                               | 1-6 days (estimated)                 |

The octanol/water partition coefficient value has been estimated from the equation of Kenaga and Goring (1980) and the value of water solubility data. Similarly, the value for the BCF has been estimated from the equation of Veith et al. (1979) and the estimated octanol/water partition coefficient value.

The half-lives of 1,2-t-dichloroethylene, due to its volatilization from aquatic media, have been estimated from the reaction rate ratio (0.601) and the oxygen reaeration rate of  $0.19-0.96 \text{ day}^{-1}$  (Mabey et al., 1981).

Pertinent data regarding the fate of 1,2-t-dichloroethylene in soil could not be located in the available literature. Based on the behavior of this compound in aquatic media, evaporation is expected to be the predominant loss process from the soil surface. In subsurface soil, biodegradation of this compound is likely to be a slow process (Tabak et al., 1981), and the compound is not likely to be sorbed strongly to soil because of its low

octanol/water partition coefficient value. Therefore, the compound is expected to leach from subsurface soil into groundwater. Page (1981) reported 1,2-t-dichloroethylene in groundwaters from New Jersey at a frequency of 51%.

## 2. ABSORPTION FACTORS IN HUMANS AND EXPERIMENTAL ANIMALS

### 2.1. ORAL

Pertinent data regarding the oral absorption of 1,2-t-dichloroethylene could not be located in the available literature. The U.S. EPA (1980a) estimates that "virtually 100 percent of ingested DCE may be absorbed systemically," based on the studies of Daniel (1963) and Monster et al. (1976) using trichloroethylene.

### 2.2. INHALATION

Pertinent data regarding the absorption of 1,2-t-dichloroethylene from the respiratory tract could not be located in the available literature. The U.S. EPA (1980a) estimates that "35-50 percent of inhaled DCE.....may be absorbed systemically," based on the studies of Daniel (1963) and Monster et al. (1976) using trichloroethylene.

### 3. TOXICITY IN HUMANS AND EXPERIMENTAL ANIMALS

#### 3.1. SUBCHRONIC

3.1.1. Oral. Springer (1965) administered a mixture of 1,2-c- and 1,2-t-dichloroethylene to rats for 7 weeks (0.05, 0.25, 0.5 or 1.0 g/kg). It was not clear from the review if these were daily, weekly or total doses. No adverse effects were reported at any dose. Pertinent data regarding the subchronic oral toxicity of pure 1,2-t-dichloroethylene were not located in the available literature.

3.1.2. Inhalation. In an unpublished study, Torkelson (1965) exposed rats, rabbits, guinea pigs and dogs to atmospheres containing 60% 1,2-c- and 40% 1,2-t-dichloroethylene (500 or 1000 ppm) 7 hours/day, 5 days/week for 6 months. No adverse effects were observed on growth, mortality, organ weights, body weight, hematology, clinical chemistry, gross pathology or histopathology.

Freundt et al. (1977) exposed groups of six female Wistar rats to atmospheres containing 200 ppm 1,2-t-dichloroethylene 8 hours/day, 5 days/week for 1, 2, 8 or 16 weeks. The liver and lungs were examined histologically at the end of each of these time periods. Progressive damage to the lungs and fatty changes in the liver were observed. By 16 weeks, 3/6 animals developed "severe" fatty changes in the liver, and 2/6 developed "slight" fatty changes.

#### 3.2. CHRONIC

3.2.1. Oral. Pertinent data regarding the chronic oral toxicity of 1,2-t-dichloroethylene were not located in the available literature.

3.2.2. Inhalation. Pertinent data regarding the chronic inhalation toxicity of 1,2-t-dichloroethylene were not located in the available literature.

### 3.3. TERATOGENICITY AND OTHER REPRODUCTIVE EFFECTS

3.3.1. Oral. Pertinent data regarding the teratogenicity or other reproductive effects of 1,2-t-dichloroethylene could not be located in the available literature.

3.3.2. Inhalation. Pertinent data regarding the teratogenicity or other reproductive effects of 1,2-t-dichloroethylene could not be located in the available literature.

### 3.4. TOXICANT INTERACTIONS

Pertinent data regarding the interaction of 1,2-t-dichloroethylene with other toxicants could not be located in the available literature.

#### 4. CARCINOGENICITY

##### 4.1. HUMAN DATA

4.1.1. Oral. Pertinent data regarding the carcinogenicity of orally administered 1,2-t-dichloroethylene in humans could not be located in the available literature.

4.1.2. Inhalation. Pertinent data regarding the carcinogenicity of inhaled 1,2-t-dichloroethylene in humans could not be located in the available literature.

##### 4.2. BIOASSAYS

4.2.1. Oral. Pertinent data regarding the carcinogenicity of orally administered 1,2-t-dichloroethylene in experimental animals could not be located in the available literature.

4.2.2. Inhalation. Pertinent data regarding the carcinogenicity of inhaled 1,2-t-dichloroethylene in experimental animals could not be located in the available literature.

##### 4.3. OTHER RELEVANT DATA

Greim et al. (1975) reported negative results for 1,2-t-dichloroethylene using Escherchia coli K12 as the indicator organism. Cerna and Kypenova (1977) found 1,2-t-dichloroethylene to be nonmutagenic in Salmonella tester strains, and this compound failed to induce chromosomal aberrations as indicated by cytogenic analysis of bone marrow cells in mice following repeated i.p. injections.

##### 4.4. WEIGHT OF EVIDENCE

Pertinent data regarding the carcinogenicity of 1,2-t-dichloroethylene in humans or animals could not be located in the available literature. Using the criteria for evaluating the overall weight of evidence for carcinogenicity to humans proposed by the Carcinogen Assessment Group of the

U.S. EPA (Federal Register, 1984), 1,2-t-dichloroethylene is most appropriately designated a Group D - Not Classified chemical.

## 5. REGULATORY STANDARDS AND CRITERIA

The ACGIH (1980, 1981) has established a TLV of 200 ppm (~790 mg/m<sup>3</sup>) and a STEL of 250 ppm (~1000 mg/m<sup>3</sup>), based upon the unpublished study by Torkelson (1965). This standard does not distinguish between the cis and trans isomers.

## 6. RISK ASSESSMENT

### 6.1. ACCEPTABLE INTAKE SUBCHRONIC (AIS)

6.1.1. Oral. The available data were inadequate for the derivation of a subchronic oral AIS for 1,2-t-dichloroethylene.

6.1.2. Inhalation. Two studies (Torkelson, 1965; Freundt et al., 1977) were located in which 1,2-dichloroethylene was administered to experimental animals. Torkelson (1965) observed no effects of subchronic inhalation exposure to 1000 ppm of a mixture of 1,2-dichloroethylene isomers (~400 ppm of the trans isomer) for 6 months. In contrast, Freundt et al. (1977) reported fatty changes in the liver and minor changes in the lungs following exposure to 200 ppm 1,2-t-dichloroethylene for 16 weeks. Because of these contradictions, the data are not considered suitable for the derivation of an AIS.

A CS was calculated for the effects of fatty liver changes and progressive pulmonary histopathological lesions observed by Freundt et al. (1977) in rats exposed to 200 ppm 1,2-t-dichloroethylene 8 hours/day, 5 days/week for 16 weeks. A human MED was obtained by expanding to continuous exposure and applying the assumptions that the human inhalation rate is 20 m<sup>3</sup> of air/day with an absorption factor of 0.5. Additionally, an uncertainty factor of 10 was applied because the Freundt et al. (1977) study was subchronic in duration. The resulting human MED of 189 mg/day corresponds to an  $RV_d$  of 2.1. The histopathologic changes in the liver and lungs were rated an  $RV_e$  of 5. A CS of 10.5, the product of  $RV_d$  and  $RV_e$ , was derived.

### 6.2. ACCEPTABLE INTAKE CHRONIC (AIC)

6.2.1. Oral. The available data were inadequate for the derivation of an oral AIC 1,2-t-dichloroethylene.

6.2.2. Inhalation. The available data were inadequate for the derivation of a chronic inhalation AIC for 1,2-t-dichloroethylene.

6.3. CARCINOGENIC POTENCY ( $q_1^*$ )

6.3.1. Oral. Pertinent data regarding the carcinogenicity of 1,2-t-dichloroethylene following oral exposure could not be located in the available literature. Therefore, no  $q_1^*$  could be derived.

6.3.2. Inhalation. Pertinent data regarding the inhalation carcinogenicity of 1,2-t-dichloroethylene could not be located in the available literature. Therefore, no  $q_1^*$  could be derived.

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# APPENDIX

## Summary Table for 1,2-t-Dichloroethylene

| Species                 | Experimental Dose/Exposure | Effect                                                                | Acceptable Intake (AIS or AIC) | Reference            |
|-------------------------|----------------------------|-----------------------------------------------------------------------|--------------------------------|----------------------|
| Inhalation              |                            |                                                                       |                                |                      |
| AIS                     |                            |                                                                       | ND                             | ND                   |
| AIC                     |                            |                                                                       | ND                             | ND                   |
| Maximum composite score | rat                        | 200 ppm 8 hours/day, 5 days/week for 16 weeks (RV <sub>D</sub> = 2.1) | 10.5                           | Freundt et al., 1977 |
| Oral                    |                            |                                                                       |                                |                      |
| AIS                     |                            |                                                                       | ND                             | ND                   |
| AIC                     |                            |                                                                       | ND                             | ND                   |

ND = Not derived